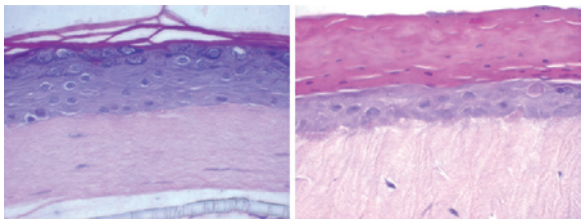
Dioxin Disses Keratinocytes

2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is an environmental pollutant that causes chloracne, a severe skin disease resulting in disfiguring scarring. Geusau and coworkers studied TCDD's effect on skin equivalents of normal epidermal keratinocytes under conditions inducing differentiation. The most prominent effects included a thicker stratum corneum with parakeratosis and loss of the granular layer. Thus, TCDD initially accelerates differentiation, demonstrated by chloracne. In the final steps of keratinocyte differentiation, TCDD reduces proenzyme caspase -14 expression and activation and completely blocks processing of filaggrin. This obstructs regular completion of differentiation, probably leading to the parakeratosis and loss of the granular layer. This accelerating differentiation *in vitro* recapitulated *in vivo* chloracne. J Invest Dermatol 124:275–277, 2005.



Mad Dogs and Englishmen

Nevus number, largely under genetic control, correlates with melanoma risk. Wachsmuth and colleagues studied 221 teenage twin pairs and concluded that 66% of the nevus count is due to genetic effects; of the 25% of variation due to environmental influences, one third is estimated to result from sun exposure. Though not able to demonstrate a significant protective effect for either sun protection cream or shirt-wearing in reducing number of nevi—maybe because high SPF or beach-type clothes allow longer exposure to UVA rays—they recommend avoiding mid-day sun as the best skin care defense. J Invest Dermatol 124:56–62, 2005.

Patch Work

The development of severe, extensive non-melanoma skin cancer is a common complication of drugs prescribed after organ transplantation, especially in patients of European descent. A new model system demonstrates the role of immunosuppression in tumors. Vogt and coworkers treated Cs137-irradiated *Ptch1*+/−mice with cyclosporine A plus prednisolone for 4.5 months; the basal cell carcinoma (BCC) burden increased by 2.5 fold. This model is useful for investigating mechanisms of drug enhancement of BCC formation (such as UV or ionizing radiation) and genetic factors that may help determine which transplant recipients may develop BCCs. J Invest Dermatol 124:263–267, 2005.

A Fish Storey

Fish oil-derived omega-3 polyunsaturated fatty acids (n-3 PUFA) inhibit UVB-induced inflammation and other inflammatory states. UVB releases interleukin-8 (IL-8), a pro-inflammatory and chemotactic cytokine, from epidermal and dermal cells. Storey and colleagues discovered that the two principal types of dietary n-3 PUFA—eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)—can reduce basal and UVB-induced IL-8 secretion by keratinocytes and fibroblasts *in vitro*. These n-3PUFAs can also suppress tumor necrosis factor-α (TNF-α)-induced IL-8 secretion in keratinocytes. Further exploring these mechanisms may lead to using n-3PUFAs as photoprotective and anti-inflammatory modulators for a wide range of conditions including sunburn, photoaging, immunosuppression, and melanoma. Hopefully, preparations will not smell fishy. J Invest Dermatol 124:248–255, 2005.